

**Amendments to the Specification**

The additions have been indicated by underlining (underlining). The deletions have been indicated by strikethrough (~~striketthrough~~).

**Please replace the paragraph on page 2 lines 16-31 with the following amended paragraph:**

Within the context of the present invention it has been found that the expression of a family of genes called plasticity-related genes (PRGs) overcomes the repellent effect of bioactive lipid phosphates, in particular of LPA and, thus, allows the regrowth of axons in spite of the presence of bioactive lipid phosphates. Therefore, the present invention is directed at an isolated protein comprising the same or substantially the same amino acid sequence selected from the group consisting of human PRG-1 [SEQ ID NO:1], human PRG-2 [SEQ ID NO:2], human PRG-3 [SEQ ID NO:3], human PRG-4 [SEQ ID NO:4], mouse PRG-1 [SEQ ID NO:5], mouse PRG-2 [SEQ ID NO:6], mouse PRG-3 [SEQ ID NO:7], mouse PRG-4 [SEQ ID NO: 8], rat PRG-1 [SEQ ID NO:9], rat PRG-2 [SEQ ID NO:10], rat PRG-3 [SEQ ID NO:11], and rat PRG-4 [SEQ ID NO: 12] (~~depicted in SEQ ID NOs: 1 to 12~~), respectively, or a splice variant or a salt thereof. A protein having substantially the same amino acid sequence comprises proteins with at least about 95%, preferably at least about 96%, more preferably at least about 97%, more preferably with at least about 98% and most preferably with at least about 99% amino acid sequence identity. The amino acid exchanges are preferably so called conservative changes meaning substitutions of, for example, a polar amino acid residue by another polar amino acid residue, of a acidic amino acid residue by another acidic amino acid residue or of a basic amino acid residue by another basic amino acid residue.

**Please replace the paragraph on page 4 lines 24-28 with the following amended paragraph:**

In a preferred embodiment of the nucleic acid of the present invention the nucleic acid comprises a nucleic acid selected from the group consisting of the human PRG-1

gene [SEQ ID NO:13], the human PRG-2 gene [SEQ ID NO:14], the human PRG-3 [SEQ ID NO:15], the human PRG-4 [SEQ ID NO:16], the mouse PRG-1 gene [SEQ ID NO:17], the mouse PRG-2 gene [SEQ ID NO:18], the mouse PRG-3 [SEQ ID NO:19], the mouse PRG-4 [SEQ ID NO:20], the rat PRG-1 [SEQ ID NO:21], the rat PRG-2 [SEQ ID NO:22], the rat PRG-3 [SEQ ID NO:23], and the rat PRG-4 gene [SEQ ID NO:24] (see SEQ ID NOs: 13 to 24).

**Please replace the paragraph which spans page 14 lines 33-34 through page 15 lines 1-6 with the following amended paragraph:**

Fig. 1 Panel A depicts the human PRG-1 amino acid sequence (SEQ ID NO. 1). The first 300 amino acids are highly conserved among LPP family members. The other 400 amino acids (gray boxed sequence) of PRG-1 show no homologies to known sequences. The catalytic histidine (His-252) is marked with an asterisk. Panel B depicts rPRG-3 [nucleic acid: SEQ ID NO:23; amino acid: SEQ ID NO:11] (~~SEQ ID NO. 11~~). Start and stop codons are marked with a dark box. Putative transmembrane domains are underlined in grey, the C-terminal tail is indicated light grey. The probes, which were used for *in situ* hybridization are marked with a black line.